735

BUREAU OF SUGAR EXPERIMENT STATIONS

BRISBANE

QUEENSLAND, AUSTRALIA

TECHNICAL COMMUNICATIONS.

YEAR 1958, No. 1

A Miscellany of Sugar-Cane Diseases.

by

C. G. HUGHES

Issued by Direction of the Sugar
Experiment Stations Board

NSP
RAM
MT

COMMONWEALTH MYCOLOGICAL INSTITUTE
5 JAN 1959

SUGAR EXPERIMENT STATIONS BOARD

THE HON. O. O. MADSEN, M.L.A., Minister for Agriculture and Stock, Chairman.
DR. W. A. T. SUMMERVILLE, Under-Secretary for Agriculture and Stock, Deputy Chairman.
MR. J. W. INVERARITY, representing the manufacturers of cane sugar.
MR. L. G. SCOTNEY, representing the growers of sugar cane.

BUREAU OF SUGAR EXPERIMENT STATIONS

N. J. KING, Director.
L. G. VALLANCE, Asst. Director.
R. W. MUNGOMERY, Asst. to Director.

Head Office Staff

J. H. NICKLIN, Chief Technologist (Engr.).
C. G. HUGHES, Senior Pathologist (I).
D. R. L. STEINDL, Senior Pathologist (II).
L. R. BRAIN, Senior Mill Technologist (II).
H. E. YOUNG, Senior Physiologist (II).
O. W. STURGESS, Pathologist.
K. C. LEVERINGTON, Chemist.
R. F. BEALE, Asst. Engineer.
A. D. DOOLAN, Laboratory Technologist.
J. R. BURGE, Analyst.
J. M. SEDL, Analyst.
C. C. HORNE, Secretary.
A. D. BEAL, Asst. Accountant.
S. C. LEDDY, Records Clerk.
E. J. BARKE, Clerk.
B. HJELM, Clerk.
MISS F. M. DOOLAN, Librarian.
MISS E. O'SULLIVAN, Clerk.
MISS J. E. MURPHY, Clerk-Typiste.
MISS B. A. MURPHY, Clerk-Typiste.
MISS H. R. PICKETT, Clerk-Typiste.
MISS J. M. MANNION, Clerk-Typiste.
F. SHORT, Lab. Attendant.

Field Staff

Meringa.

J. H. BUZACOTT, Senior Plant Breeder (I).
G. WILSON, Senior Entomologist (II).
J. C. SKINNER, Senior Geneticist (II).
B. G. ADKINS, Mill Technologist.
B. T. EGAN, Pathologist.
B. E. HITCHCOCK, Entomologist.
G. BATES, Senior Adviser.
A. G. BARRIE, Asst. Agronomist.
C. A. REHBEIN, Adviser.
N. G. GRAFF, Technical Officer (I).
F. C. LINDSAY, Technical Officer (I).
G. H. WHITAKER, Field Assistant.
P. G. ARNOLD, Cadet.
MISS N. J. SMITH, Clerk-Typiste.
MISS M. BARBER, Clerk-Typiste.
R. C. MCALLOON, Lab. Attendant.

Innisfail.

S. O. SKINNER, Senior Adviser.
I. T. FRESHWATER, Field Assistant.

Ingham.

O. W. D. MYATT, Senior Adviser.
I. J. V. STEWART, Field Assistant.

Ayr.

G. A. CHRISTIE, Senior Adviser.
J. WESDORP, Asst. Agronomist.
L. S. CHAPMAN, Adviser.
J. ANDERSON, Adviser.
MISS B. L. LENNOX, Clerk-Typiste.

Mackay.

G. C. BIESKE, Chemist.
C. G. STORY, Senior Adviser.
A. A. MATTHEWS, Adviser.
C. M. MCALEESE, Adviser.
L. E. RODMAN, Field Assistant.
J. E. MILLIKEN, Field Assistant.
MISS J. L. CRONIN, Clerk-Typiste.
MISS C. HARVISON, Clerk-Typiste.
J. VELLA, Lab. Attendant.

Bundaberg.

R. DEICKE, Senior Mill Technologist (II).
A. G. CLAIRE, Mill Technologist.
R. B. MOLLER, Agronomist.
H. G. KNUST, Senior Adviser.
N. MCD. SMITH, Senior Adviser.
E. D. JENSEN, Asst. Mill Technologist.
F. GALETTO, Asst. Mill Technologist.
V. V. JENKINS, Adviser.
R. K. HILHORST, Technical Officer (II).
J. A. HUCKNALL, Field Assistant.
MISS J. PRIEBE, Clerk-Typiste.

Nambour.

C. L. TOOHEY, Adviser.

BUREAU OF SUGAR EXPERIMENT STATIONS
BRISBANE
QUEENSLAND, AUSTRALIA

TECHNICAL COMMUNICATIONS.

YEAR 1958, No. 1

A Miscellany of Sugar-Cane Diseases.

by
C. G. HUGHES

Issued by Direction of the Sugar
Experiment Stations Board

A Miscellany of Sugar-Cane Diseases

By C. G. HUGHES

INTRODUCTION.

The Bureau Pathology Farm (Hughes, 1953) is well isolated from commercial cane plantings and so can be safely used for work on diseases which are not present on the farmers' assigned areas, or are present in such small amounts that any deliberate multiplication for any purpose would be inadvisable. Resistance trials for diseases such as leaf scald, Fiji, downy mildew, red rot and gumming therefore are conducted on the Farm and form a major part of the activities there. However, experimental work on several diseases is also undertaken and there is a most interesting section used as a living museum for the observation of stools affected with unidentified diseases. The term "disease" is here used in its widest sense, for in many instances, the so-called diseased stool turns out to have been the victim of the external environment on its original site, rather than to have suffered from any pathological condition: in other instances, genetical causes are obviously to blame.

It is the policy of the Pathology Division to encourage all Bureau officers and all officers of the Cane Pest and Disease Control Boards (Hughes, 1950) to forward specimens of diseased cane, preferably including stool pieces or other planting material, to the Farm. Individual farmers are also very often disease conscious and themselves assist officers in locating the odd and the abnormal in their crops. The Board officers in the various districts maintain a constant patrol of the growing crop until it is too large for inspection and, without a doubt, Queensland cane fields are under more constant surveillance than those of any other country. Inspectors know when anything is outside their experience and a continual stream of specimens is sent to the pathologists for examination and report. In some instances the items may be easily identified but often the specimens, after microbiological examination, have to be planted at the Farm for further observation. Some grow away into normal plants without any further sign of disease but those affections which are sett transmitted and are not lethal, eventually become established at the Farm. They are then propagated in small plantings for observation, and a constant watch kept to see that there is no undue spread from them.

It should be mentioned that even though the Farm is well isolated, there is never any attempt made to maintain there any abnormality showing in a cane being imported from another country. Such items, pathologically fascinating though they be, are destroyed while in quarantine without any attempts at isolation of a possible causal agent. All affected canes receive the same treatment whether they come from recognized producing countries or from wild areas such as New Guinea.

The collection of specimens from Queensland cane fields has been going on for some years and the museum now contains an interesting array of sett-transmitted conditions, some of which appear to be caused by pathogens, others of which could well be genetical in origin. Several of the diseases have been seen frequently since

first reported, *e.g.* dwarf, bacterial mottle and sclerophthora, but there remain a number which, often occurring in only one or a few stools, have not become established as recognized diseases of sugar cane. They could well form subjects for study for a higher degree by a graduate research student, but their present economic importance does not warrant any specific attempt at a thorough investigation.

Brief descriptions and notes on a number of the diseases currently growing in this miscellaneous section at the Farm are included below. However, common genetical effects such as sectorial chimeras and albinisms are not discussed, although mention is made of some aberrant forms with a gross distortion of habit.

The lack of information on the etiology of the diseases described makes it very difficult to suggest names which will stand the test of time. It is proposed, therefore, to refrain from attempting to name the conditions specifically and they will be referred to only in general terms.

MISCELLANEOUS DISEASES.

A Disease of Q.49.

Pokkah boeng (Bell, 1929) is such a common disease in Queensland, particularly with the first flush of new growth at the commencement of the wet season, that usually little attention is paid to the stalks showing the characteristic mutilated

FIG. 1—Malformation of the young shoots of Q.49.

tops. In 1952, however, in a field of first ratoon Q.49 at South Bingera in the Bundaberg district, a number of stools became conspicuous by showing symptoms similar to those of pokkah boeng in a severe form when the rest of the field did not show any at all. The distribution of the diseased stools indicated that the setts from which they came had been diseased when planted, for the diseased stools were in groups along the row corresponding to stick lengths dropped by the cutter planter. This sett transmission was confirmed by the discovery of the disease in the older ratoons which had been used as a source of plants. The systemic infection, of course, precluded the disease being pokkah boeng.

Plantings and ratooning have been continued at the Farm since the initial planting in 1952 and the disease is still maintained there, although on at least two occasions it appeared as though the disease had been lost. In each instance, however, the disease showed again in the wet season, even when the stools had behaved perfectly normally for the first few months.

Setts from diseased stalks may give rise to normal leaves, but usually some malformation is noticeable soon after the shoots first emerge (Fig. 1). Growth is slow and stooling delayed. The spindle is often chlorotic with very much shortened leaves and the older, longer leaves have an abnormal harshness even when almost normal in habit. More badly affected leaves are very short and erect, and show a cracking of the necrotic leaf edges. A diseased stool commonly shows both diseased and healthy shoots. As the stool grows the diseased shoots are quickly overshadowed and some may die, but some remain alive and continue to display some remarkable abnormalities. The top may suffer damage to a varying degree and at times may have the typical appearance of a shoot affected with Fiji disease (Fig. 2); the leaves are dark in colour, harsh, erect and shortened, but the typical leaf galls of Fiji are of course absent. Other galls sometimes appear in abundance on the stem and on the leaf sheaths (Fig. 3), and there may be some adventitious shooting of the buds. At times, the shoots lose their capacity to respond to gravity and will grow in a curve without the usual attempt to regain the vertical. The bending commences in the fully formed, mature portion of the stem and gives the impression that the top has suddenly become too heavy for the stalk to support. The bent stalks show wide growth rings—up to one inch in width — somewhat swollen root

FIG. 2— Shoots of Q.49 affected by the unnamed disease are quite malformed.

bands, and cankering in the region of the nodes. Later the membrane covering the tyre-like, swollen root band ruptures and the leaf sheath, instead of becoming detached at the scar below the node, breaks off at the level of the root band, leaving a ring of dead sheath tissue still attached. All except the older internodes show a number of rind galls and occasional necrotic areas; the smooth enlarged growth rings are largely free from galls. The upper nodes are blackened and rotting, with or without a weft of mycelium, and often show spectacular, broad knife cuts in the region of the growth ring (*see* Fig. 3).

The overall appearance of the disease suggests that a hormone spray may have been applied to excess, but in no instance since the discovery of the disease has any hormone been brought into contact with the affected plants.

FIG. 3—The disease in Q.49 causes malformation, cankers and galls on the leaf sheath.

The symptoms of the disease in some ways resemble those due to spike disease (Sharma and Jha, 1957) and both reach their best development in the wet season. Sharma and Jha, however, do not record the extensive galls, nor the hypertrophied root band, which is such a feature of the disease under discussion. The differences may be a varietal effect, and it is possible that the two diseases are the same.

A Disease of N.Co.310.

In March, 1952, a markedly stunted stool of N.Co.310 was observed in a field on the Mackay Sugar Experiment Station. It was dug out and forwarded for planting at the Pathology Farm. The drying of the leaf tissue en route prevented a critical examination at the time but within four months or so the new growth from the planted stool left no doubt that a new cane disease had been found. Further inspections at Mackay have failed to reveal any other affected stools although as this communication goes to press comes a report of several stools in the Rocky Point area between Brisbane and the New South Wales-Queensland border.

Plantings have been made using the tiny setts, which are all that are ever produced, and the disease has continued to be propagated without difficulty at the Farm.

At first it was thought that the disease may have been sembur (Hes, 1953) but subsequent discussions with Hes and further experience with this new disease, brought to light marked differences between the two diseases and it is now considered that they are quite distinct.

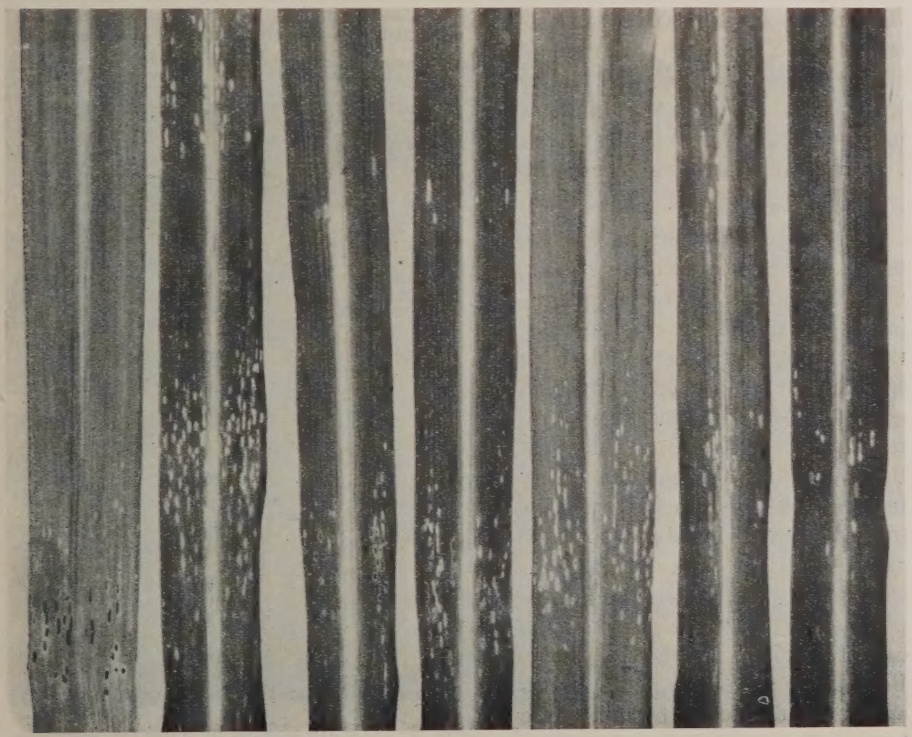

FIG. 4—Disease in N.Co.310. The banded arrangement of lesions on the young leaves is most apparent during the summer growing period.

Planting material from diseased stools germinates quite satisfactorily although symptoms show at a very early stage. Leaves on the young shoots usually show only a few lesions in well marked discrete groups, often with only one such group on each blade (Fig. 4). The symptoms consist of yellowish or necrotic brown spots extending in a diffuse band perhaps 3-4 cm. wide across the leaf blade and including the mid-rib. The spots are squarish or rectangular in outline and average about 1 x 2 mm., with the long axis in the direction of the venation. The yellowish spots do not show any damage to the leaf except the discoloration but, in the necrotic areas, the leaf tissue is thinner than normal and may be broken away in an open crack. Yellow spots on the mid-rib are much the same as on the blade, but spots with necrosis show a marked ladder-like lesion with brown dead tissue lining a narrow cavity. These ladder lesions are very similar to those caused by pokkah boeng disease (Bell, 1929).

The relative position of the bands of lesions on successive leaves indicates that some factor operating in the spindle is responsible for the symptoms and dissection of the spindle reveals lesions occurring according to the same pattern, through several layers of rolled leaf tissue. These lesions are rusty brown in colour, when they show some blocking of the sub-stomatal cavities and/or dark necrosis, or else are quite colourless and transparent and obviously caused by a complete collapse of the leaf tissue. Both these types of spindle lesions apparently develop into the necrotic type on the open leaf, but those that

FIG. 5—Shortened leaves, fan-like top and spotted blad s are characteristic of a disease in N.Co.310 in winter.

FIG. 6—N.Co.310 affected by a disease described in the text, shows a marked change in habit.

become the yellow type in which the leaf surfaces remain intact, are discernible only with difficulty in the rolled spindle.

The first few leaves produced are usually quite normal except for the spots, but within a few weeks the new leaves produced are shortened, and more harsh and erect than normal. This gives a marked fan-like effect to the top (Fig. 5). Subsequent development is very slow and the total height of the diseased shoots never exceeds about three feet or so, even with continuous adequate water and fertilizer. All stalks in the stools are affected and to date there has never been any recovery. Stooling is not adversely affected, if anything it is somewhat stimulated, and the general appearance after some months of growth is of a dense, low hedge (Fig. 6).

There has been no opportunity for transmission studies but diseased stools have been growing in close proximity to a range of varieties, including N.Co.310, for several years and yet not once has there been any secondary spread of the disease, although mosaic and Fiji disease spread fairly readily in an adjacent field.

A Possible Virus Disease of Q.13.

The variety Q.13, although never a commercial cane of any consequence, was for many years a useful standard in the seedling selection work at the Northern Experiment Station, chiefly on account of its erect habit, reasonably vigorous growth

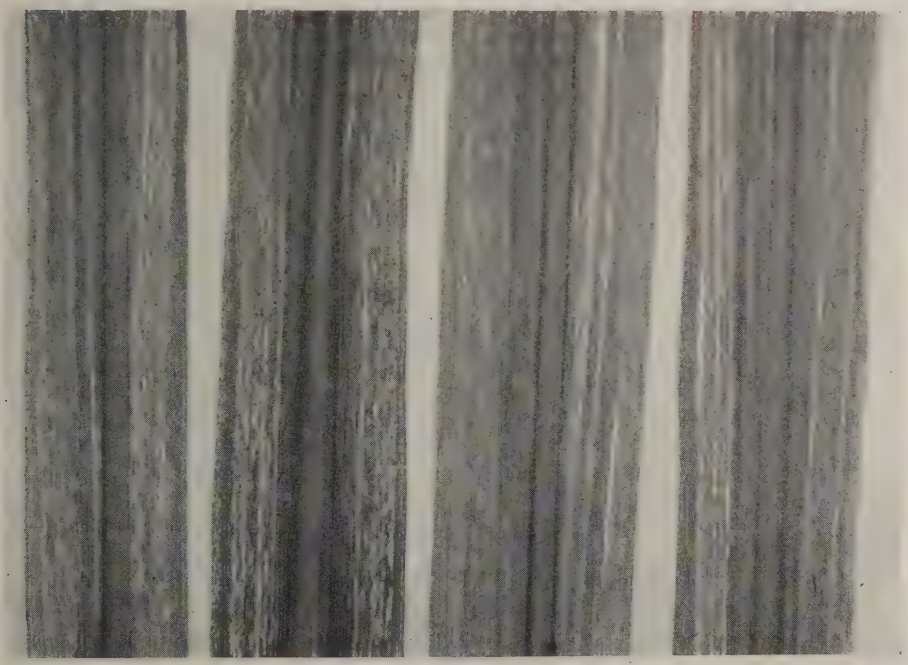

FIG. 7—A disease in Q.13 resembling streak.

and satisfactory sugar. As a standard in the seedling fields it was constantly under surveillance, and in 1945, an abnormally freckled stool was noted and subsequently transferred to Brisbane for observation. Q.13, like many other canes, usually shows a mild freckle on the leaves. It does not cause any discernible damage and, although mindful of the fact that mosaic disease in many plants was once considered as part of the nature of the variety, the probability is that this indeterminate, slight spotting is not pathological. The abnormal freckling was, however, of immediate interest, especially when it was found associated with some loss of vigour and severely cankered stems.

Germination and ratooning are not affected by the disease and the freckling appears on the first leaves as they unfold from the spindle. It shows as yellow green spots on the green of the blade and mid-rib (Fig. 7). These have much the same appearance on both surfaces and are approximately one millimetre in width with a somewhat greater length in the direction of the venation. They are usually confined within the major veins and while obvious on the very young leaves, are more pronounced on the mature and older leaves, particularly in cane which is growing rapidly. They can be very numerous and often run together to form somewhat ill-defined, mottled streaks. There is no marked concentration towards the base of the leaves and the streaks vary considerably in width. At times the pattern suggests that of mosaic, which was the reason for the testing of a transmission method successful with mosaic disease (*q.v.* below) but at other times it is suggestive of streak disease (Storey, 1925). Specimens were therefore forwarded to A. McMartin, formerly Pathologist of the Experiment Station of the South African Sugar Association, who had had considerable experience with the disease. He replied: "It is difficult, of course, to make an authoritative pronouncement without seeing the cane grow, as we do sometimes get specimens of leaf markings bearing some resemblance to streak disease, but which on planting and keeping under observation we decide is not. These specimens, however, are more like true streak than many of the suspected ones we have observed." The present author has not seen streak in the field and is unable to pass an opinion.

On the older leaves, small red spots develop in the yellow flecks: these may also coalesce to give narrow streaks up to 7 mm. long. Fungal mycelium has been seen in these necrotic areas, but it could be a secondary infection.

Cankers on the stem can be seen as soon as it starts to make cane but they reach their best development in the mature stalk. They are similar to those seen in mosaic (Bell, 1929) in general appearance. They may be grey to red-brown in colour and although they are generally shallow and so only involve the rind, some longitudinal splits occur into the underlying stem tissue. Q.13 is normally resistant to stem rots so that the splits usually remain free from infection. The cankers vary considerably in total area but in some instances internodes with over 90 per cent. of the surface involved have been noted.

Several attempts have been made to transmit the disease mechanically according to the method of Sein (Sein, 1930) by pricking through a diseased leaf wrapped tightly around the spindle. In each instance, mosaic disease inoculated concurrently into other shoots gave excellent transmission, so it is possible that the disease under

discussion is not a mosaic type. Two instances only of natural transmission are on record. They occurred in 1950 when a stool in a row of P.O.J.2878 growing some 22 yards from the diseased Q.13 produced a diseased shoot: two such shoots were also seen in the same variety in a row adjacent to the Q.13. Both were ratoon crops.

Spindle Stripe.

Although this disease has already been given a name (Hughes, 1953) the only previous reference to it occurs in an Annual Report (*ibid*) which may not be generally as accessible to pathologists as this Communication.

Spindle stripe has been reported from the Mt. Bauple and Maryborough areas in a number of plantings and in a single planting in the Bundaberg district. The variety concerned in each instance is Q.50 and several hundred stools have been

FIG. 8—Spindle stripe in Q.50 is not an obvious disease. Healthy leaf on left.

rogued from affected fields. Chief symptoms in the leaves consist of regular but not obvious streaks ranging in width from narrow striations to 3 mm. or so (Fig. 8). They are only distinguishable by their slightly paler colour than normal and, even when most obvious towards the base of the two youngest unfolded leaves, they are not easily seen; even experienced inspectors cannot expect to find every diseased stool. There is no necrosis and the older leaves do not show any symptoms.

Diseased stalks show some stunting, especially in the ratoons and are often more oval in cross section than normal. Growth cracks are more numerous and

FIG. 9—An effect in Trojan, similar to spindle stripe in Q.50, causes an alteration in habit and a severe stunting. Healthy Trojan on left, diseased on right.

severe than in normal cane, and fine vertical striations extending from growth ring to wax band occur on all internodes, except at the base of the stalk. The striations are also apparent on the leaf sheaths. There is a slight change in texture in the leaves and a waviness of the edges but the diseased tops are not obviously different.

Attempted isolations have not yielded any suspected pathogens and sections of the leaves do not show any invading organism. Diseased setts of course give rise to diseased plants.

A similar condition, which could actually be spindle stripe, has been found in the Inkerman area of the irrigated Lower Burdekin district (Fig. 9). It occurred in only a few stools of Trojan in one field. Replantings at the Pathology Farm have shown a faint, inconspicuous striping in the young leaves with a marked

reduction in vigour; the leaves generally are slightly harsher and stiffer than normal and, despite adequate fertilizer, are yellower than would be expected, often with an indistinct, mottled appearance. The leaves are markedly rolled, giving a wilted effect, even in moist soil.

It is possible that these faint stripe effects on the leaves represent one extreme of a range of stripings due to genetical aberrations, the other extreme being the marked, clear-cut white striping termed variegation. Any worker in sugar cane is familiar with the obvious examples, but it is when the symptoms become faint and ill defined that uncertainty as to their origin creeps in.

Freckles and Spots.

The amount of leaf freckling and spotting which might be considered normal, varies markedly between different varieties. Some, such as S.J.16 in Queensland show so much spotting so regularly in all plantings that it has come to be accepted as a characteristic feature of the cane. The leaf marking may be part of the genetic make-up of the variety, but, on the other hand, it is possible that the variety could be rendered free from it. This particular type of freckling is in all stocks of the variety so that it is impossible to judge whether any damage is being caused; its expression is also independent of the environment, although there is frequently some variation in amount from time to time during the growth and maturation of the crop. Some other varieties produce freckles under certain environmental

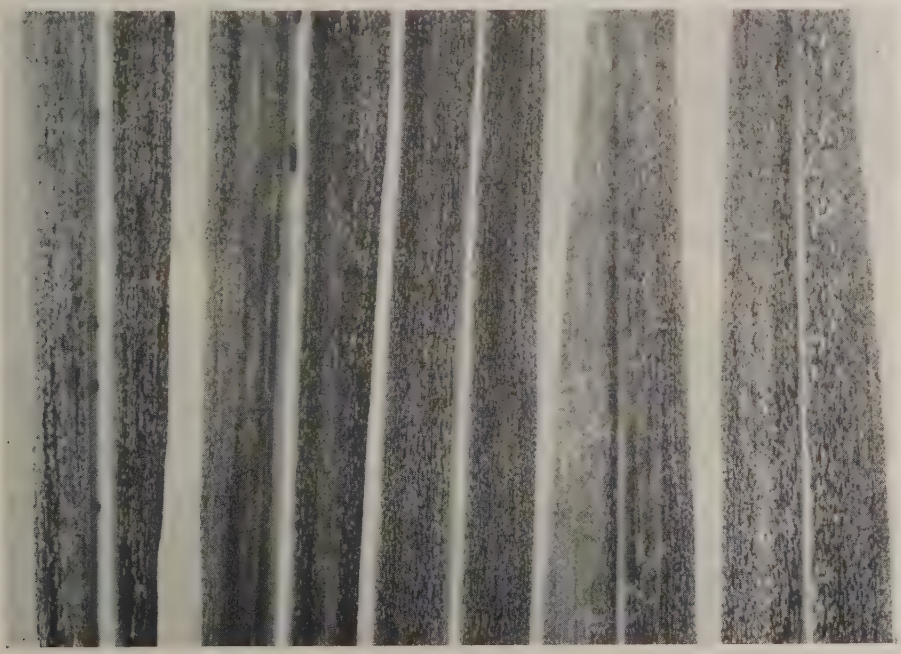

FIG. 10—A freckle in Trojan (Item 662).

conditions even when obvious agencies such as parasites, insects, mites and nutrient deficiencies are absent. The variety Q.28, for example, is liable to produce a dense red freckling on individual blades, or even parts of blades, but the conditions favouring it are not known. Setts from affected shoots do not necessarily transmit the freckling.

Apart from these two types of frecklings there are others which are sett transmitted but which occur only in a very small percentage of the stools; they are definitely not characteristic of the varieties and in some instances are obviously

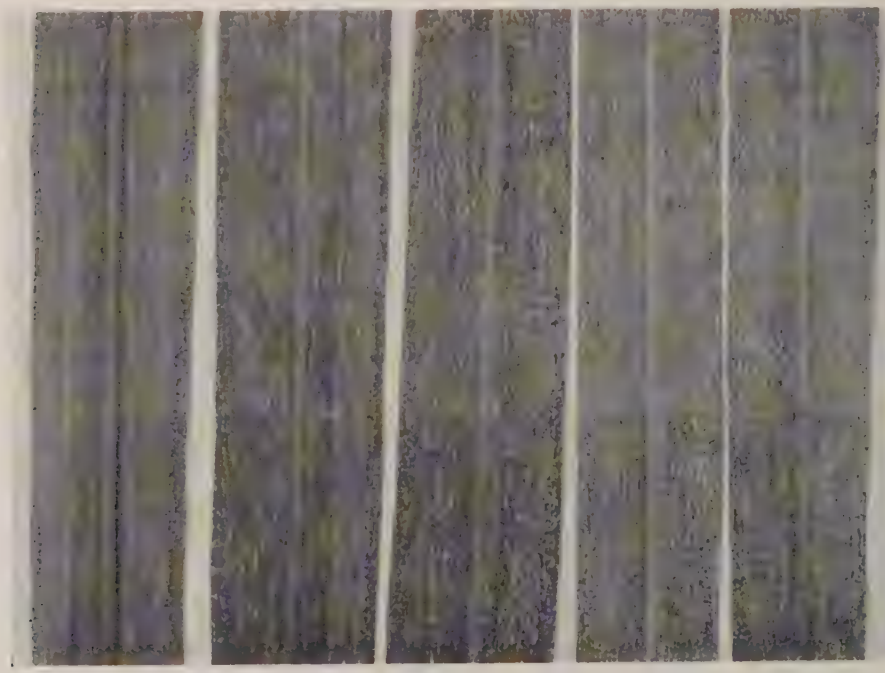

FIG. 11—A freckle in Q.50 (Item 1203).

causing some damage to the affected plants by reducing the area of effective green tissue. They are so rare that the economic effect is negligible, but their propagation should be avoided. Some of them have been under observation at the Pathology Farm and elsewhere for several years but there has been no transmission to adjacent cane.

Odd stools of Trojan in the Burdekin district, where it is easily the most important variety, have shown a distinctive freckling (Fig. 10) quite unlike anything that has been seen in other varieties (Item 662*). It shows, as the leaf unrolls, a number of light yellowish-green spots up to 1-2 mm. in length and often showing

*In the absence of specific names the Item number should help in referring to the various diseases described.

small red dots. As the leaf matures the numbers increase enormously until the greater part of the leaf area is involved. The spots on the older leaves may show a whitish colour rather than a yellowish-green and this, coupled with the small red dots, gives a pinkish-yellow to pinkish-white net-work spread over the normal green of the leaf. There is no marked necrosis and leaves do not die prematurely.

The variety Q.50, which for some years as a major cane in Central and Southern Queensland has yielded well over two million tons per annum, has shown some unusual freckling on rare occasions. One (Item 1203, Fig. 11) is a very fine freckle,

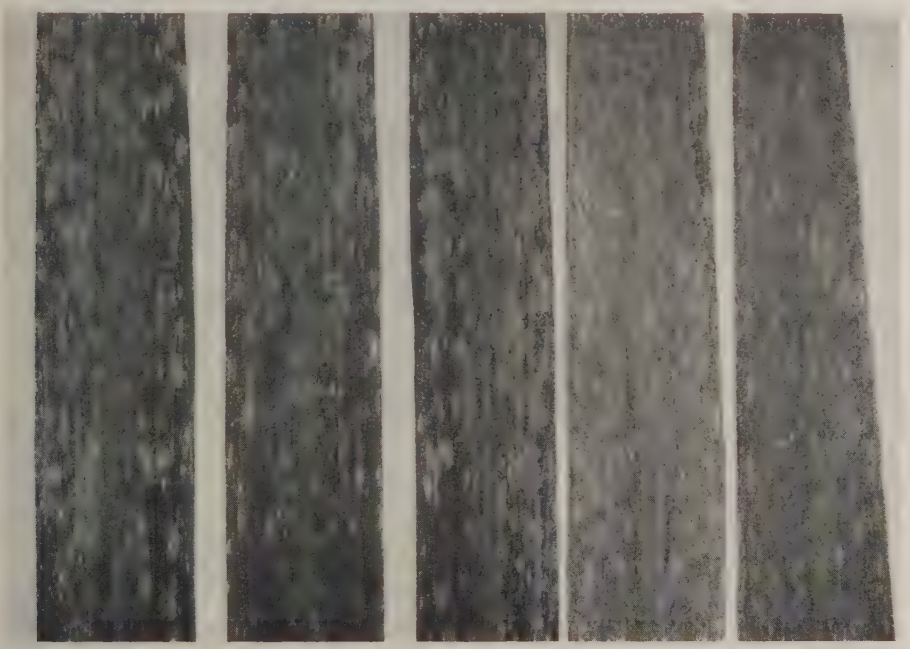

Fig. 12—Another freckle in Q.50 (Item 1207).

first showing as small yellowish-green dots on the newly unfolded leaves, and then increasing in amount without any change in colouring, until practically the whole blade is covered in the mature leaves. The spots are so numerous that these leaves assume a light yellowish-green colour, which at a distance of a few paces looks perfectly uniform. There is very little fusion of the spots and no necrosis and the leaves age and die at the usual time. The other Q.50 freckle (Item 1207, Fig. 12) also shows as small yellowish-green dots on the very young leaves, but as the numbers of freckles increase on the maturing leaves, a different pattern emerges. The spots tend to aggregate in clusters and even when practically the entire leaf blade is covered with the spots, the clusters still show as diffuse, lighter coloured areas. While growth is rapid the older leaves are a general yellow-green colour, liberally sprinkled with small red dots and with a scattering of the diffuse clusters. As the cooler weather brings a cessation of growth the leaf lesions on the older leaves show much

more colour and the red flecks in the yellowed areas become even more numerous and may fuse to form short red streaks. The clustered yellow freckling on the younger leaves often darkens to a dull liver colour.

Blotches.

The term "blotches", in contradistinction to "freckles" and "spots," may be applied to those discolorations which occur in discrete units over comparatively large areas of the blade. As in other diseases mentioned in this Communication, the leaf blotches described here are sett transmitted and occur irrespective of the environmental conditions and, of course, no pathogen has as yet been found associated with them.

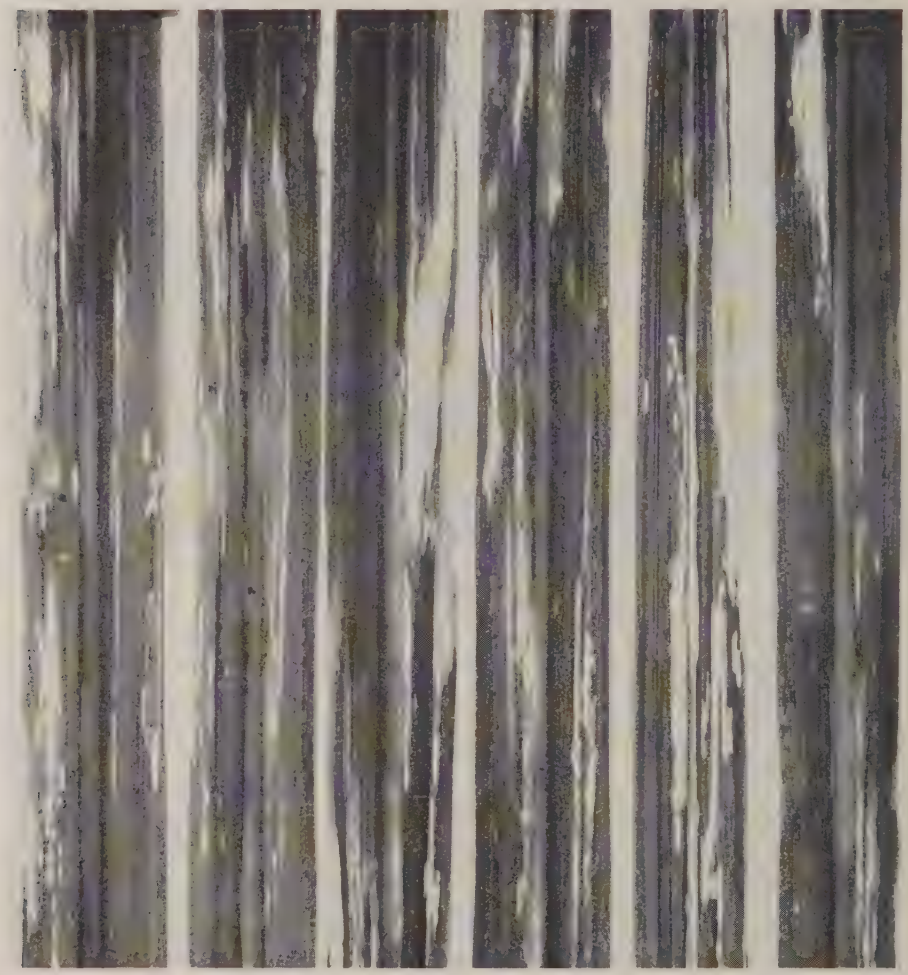

Fig. 13—Large blotches on leaves of N.Co.310 (Item 1017).

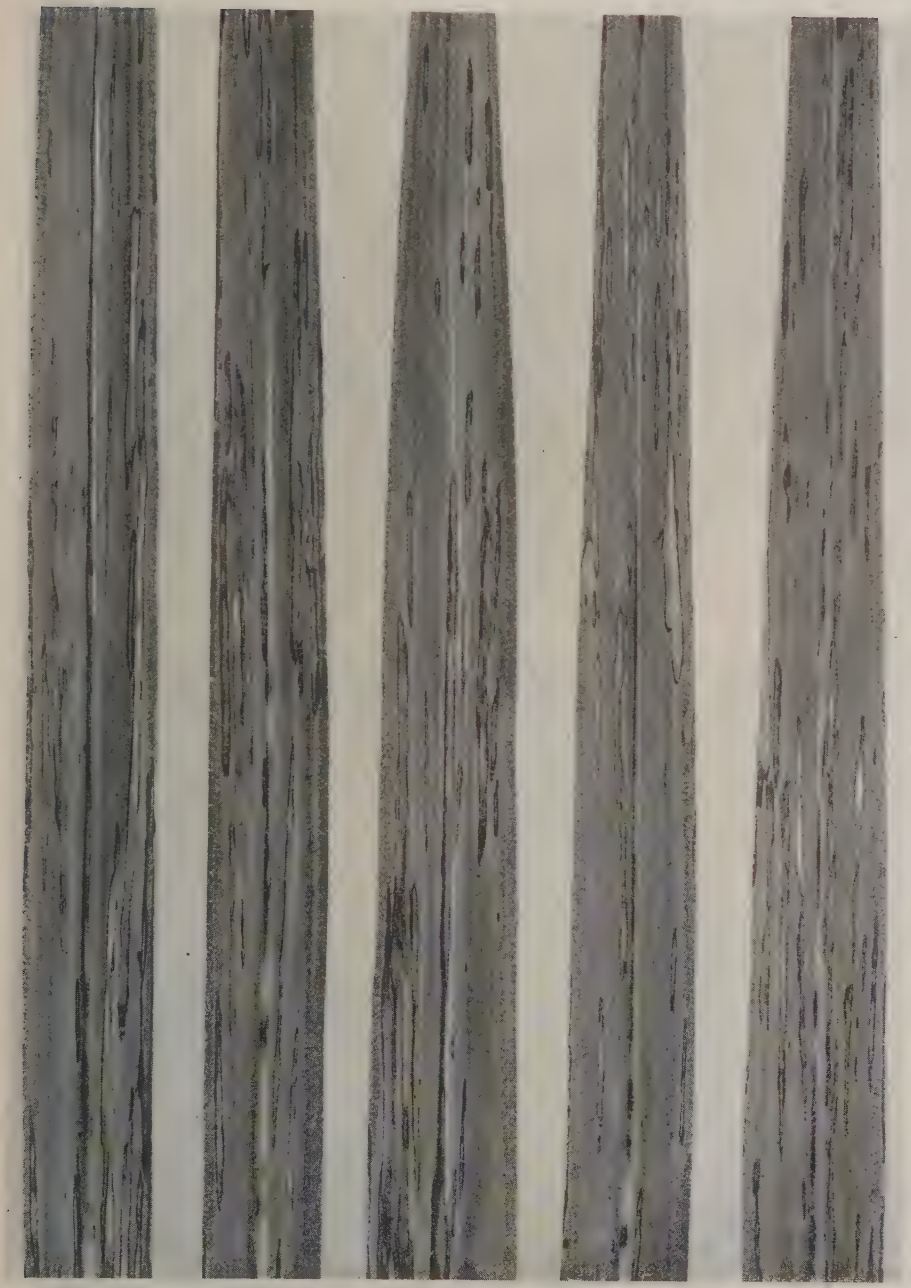

FIG. 14—Leaf blotches on N.Co.310 (Item 1205).

The variety N.Co.310 has yielded two distinct types of leaf blotches (Fig. 13 and 14). One (Item 1017, Fig. 13) is characterized by very large blotches on the leaves which are particularly noticeable during the growing season. The spindle itself may be pale with large water-soaked areas, and the unfolding leaf shows large areas of biscuit-coloured necrotic tissue edged with a narrow, red border. Up to two-thirds of this leaf may be involved but usually it is much less. Expanded leaves show irregular, yellowish, longitudinal lesions varying in size from quite small to more than half the length of the blade. In addition there are similarly shaped areas of purely necrotic tissue which later develop pronounced, narrow, red borders. The lesions do not appear to spread during the life of the leaf. The general effect of the extensive blotching is as though some caustic liquid had been thrown on to the shoot. Diseased stalks do not grow as vigorously as healthy ones and growth ceases earlier in the season. There is then a marked shortening of the leaves and a general bunching of the top; the chlorotic or necrotic lesions in the upper half of the leaves frequently cause a bowing so that the normal appearance of the top is completely lost.

The other leaf blotch in N.Co.310 (Item 1205, Fig. 14) first shows a few irregularly shaped blotches on the youngest leaf. In rapidly growing cane, they rarely exceed four in number and are up to 2 cm. in length by 2 mm. in width. There is a wide, dark reddish border surrounding an ashy centre and a pronounced halo is obvious by transmitted light. Further development occurs as the leaf expands and many lesions become apparent on the mature leaves. Individual lesions

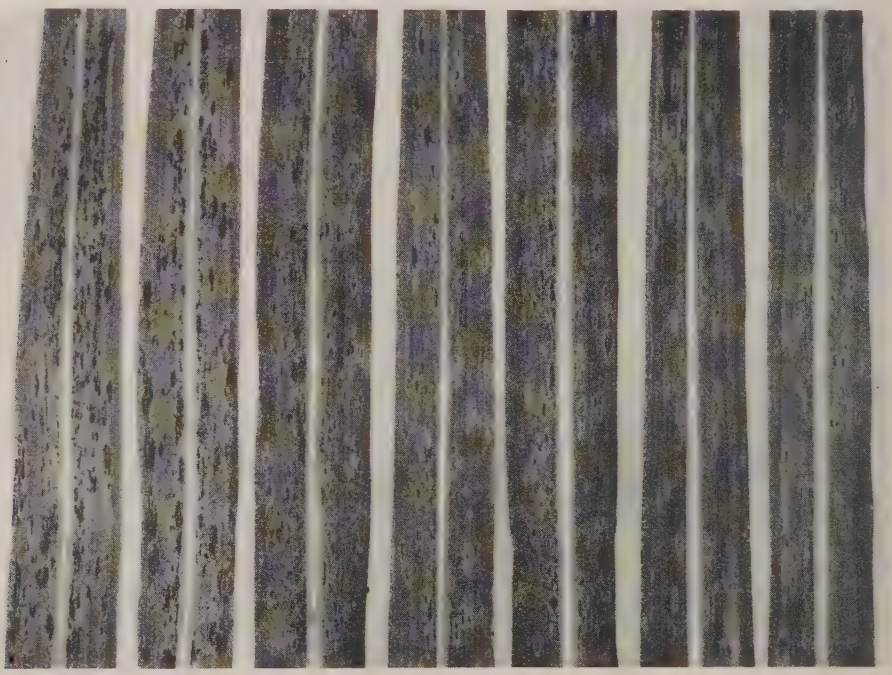

FIG. 15—A brilliantly coloured leaf blotch in Vesta (Item 972).

may be up to 25 cm. in length, but they remain comparatively narrow and rarely exceed 5 mm. in width, although some fusion of adjacent lesions may occur. The general impression given is of discrete, affected areas rather than a striped effect. Lesions may occur on any part of the blade and also on the mid-rib and along the margins of the leaf sheath. Old leaf blades become covered with fused lesions but the dark-red colour disappears and the necrotic blades are a practically normal, light biscuit shade. The older affected leaf sheaths show a similar necrosis towards the margins with some splitting. Despite the spectacular lesions on the leaves there is very little stunting of the plants and the general habit is not affected.

A single stool of Vesta in a field at Bundaberg, South Queensland, has yielded material for the propagation of an unusual, highly coloured leaf blotch (Item 972, Fig. 15). The first indication of abnormality in a diseased shoot is the production of a few, irregular yellowish markings on the first, or second, fully expanded leaf. By the third leaf the insignificant, indistinct spots have developed into mottled, bright red and yellow blotches up to 1 cm. in length and 6 mm. in width. They are scattered over the whole blade but are less numerous towards the base. With further maturation of the leaf there is an increase in number and size of the blotches and an increase in the bright-red mottled areas. Subsequently the brilliant colouring goes, to be replaced by a dark, dull red, covering most of the upper part of the blade. Leaves age normally and even at the end maintain their usual shape. The dry trash is markedly dark red in comparison with that of healthy plants. There is no marked effect on vigour.

The variety S.J.16, a minor noble cane grown in the Lower Burdekin district, has also produced a brilliant leaf affection (Item 869, Fig. 16). It first shows as

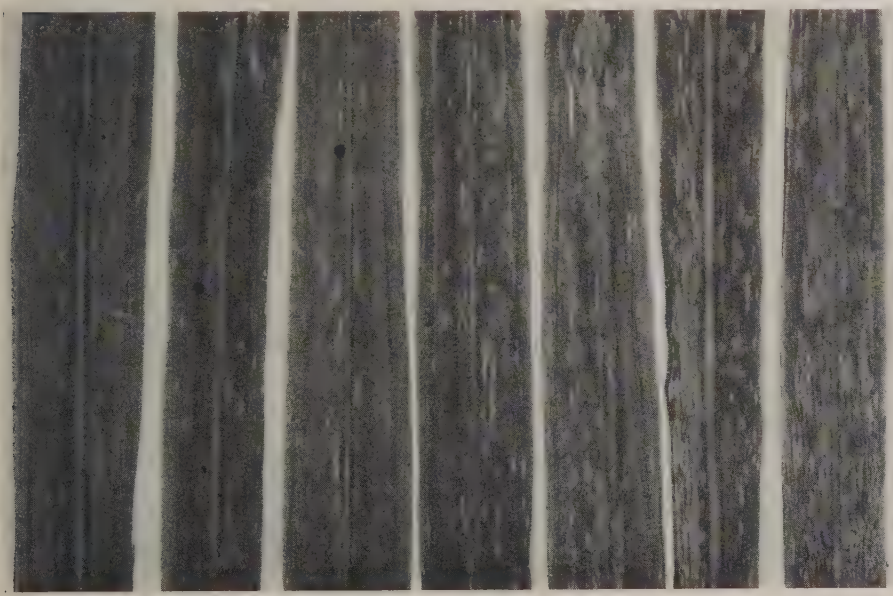

Fig. 16—A brilliant blotching of leaves of S.J.16. The youngest leaf is on the left.

small, indefinite, yellow or red spots on the young leaves which develop into small, brightly coloured areas up to a maximum of 1 cm. in length. These are clear cut and well defined, and a uniform bright tan-red in colour, sometimes with a darker border. There is a tendency for them to fuse along the venation and so give a rough striping effect. The brilliance disappears from the aged leaves but the trash is more coloured than normal. The habit of the affected shoots is not changed except that there is some overall stunting.

A Disease of Q.50 (Item 1212).

A planting of Q.50 at Mirani in the Mackay district has shown an effect which bears no resemblance to any other cane disease. Affected plants initially grow normally and are indistinguishable from their healthy neighbours until early autumn. At this time, the younger leaves develop a dull-red colour in a surge effect from the

FIG. 17—A spectacular disease showing in Q.50 in the autumn.

base of the blade upwards along the mid-rib and down into the sheath, and large chlorotic areas show on the blade. The spindle leaf is mostly chlorotic with a comparatively small amount of red. Many of the major vascular bundles in the young leaves remain green at this stage and the distal parts of the blades are quite normal. As the symptoms intensify with the approach of winter more leaves become involved and the reddening spreads on leaves already showing symptoms; in addition, it is noted that the dewlap becomes brittle and many leaves snap there, so allowing the blades to fall from their usual upright position (Fig. 17); the external spindle leaves die, but the spindle can only be pulled out with difficulty, when it is found that the necrosis involves the spindle tissues only and the growing point is still apparently sound. There is no odour associated with the necrosis and sectioned stalks are normal except for some piping. The growing point may eventually die but the stalk remains sound for some time. Some shooting of the top buds may occur in a normal fashion, except that the bud scales are reddened, and some reddening occurs at the tip of the shoot. A similar picture is presented by any suckers that may develop at this time and also by any young ratoon shoots. Such ratoons gradually lose their symptoms as the warm weather approaches, and resume normal growth during the summer, but again develop symptoms as winter approaches.

REFERENCES

Bell, A. F., 1929. A key for the field identification of sugar cane diseases. Bur. Sug. Expt. Stat., Queensland, Div. Path., Bull. 2, 23, 27-28.

Hes, J. W., 1953. Sembur leaf-spot disease of sugar cane. Inter. Soc. Sug. Cane Tech., Proc. 8th Cong., 891-894.

Hughes, C. G., 1950. The control of cane diseases in Queensland. Inter. Soc. Sugar Cane Tech., Proc. 7th Cong., 465-472.

———— 1953. The Bureau Pathology Farm. Cane Growers' Quart. Bull., Bur. Sug. Expt. Stat., Queensland, 17, 57-61.

———— 1953. Disease investigations. Miscellaneous. 53rd Ann. Rept., Bur. Sug. Expt. Stat., Queensland, 46.

Sein, F., 1930. A new mechanical method for artificially transmitting sugar-cane mosaic. J. Dept. Agric., Puerto Rico, 2, 14, 49-68.

Sharma, S. L. and Jha, J. N., 1957. "Spike" (a new) disease of sugar cane. Proc. Ind. Acad. Sciences, 45, 1 Sect. B, 16-20.

Storey, H. H., 1925. Streak disease of sugar cane. Union of South Afric., Dept. of Agric., Science Bull., 39.

Wholly set up and printed in Australia by
WATSON, FERGUSON and COMPANY
Brisbane, Q.
1958